```
 * * * * * * RECONNECTED TO STN INTERNATIONAL * * * * * *
SESSION RESUMED IN FILE 'HOME' AT 12:48:04 ON 11 JAN 2002
FILE 'HOME' ENTERED AT 12:48:04 ON 11 JAN 2002

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.15       0.15

=> file medline, biosis, embase, scisearch, caplus

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.30       0.30

FILE 'MEDLINE' ENTERED AT 12:48:32 ON 11 JAN 2002

FILE 'BIOSIS' ENTERED AT 12:48:32 ON 11 JAN 2002
COPYRIGHT (C) 2002 BIOSIS(R)

FILE 'EMBASE' ENTERED AT 12:48:32 ON 11 JAN 2002
COPYRIGHT (C) 2002 Elsevier Science B.V.  All rights reserved.

FILE 'SCISEARCH' ENTERED AT 12:48:32 ON 11 JAN 2002
COPYRIGHT (C) 2002 Institute for Scientific Information (ISI) (R)

FILE 'CAPLUS' ENTERED AT 12:48:32 ON 11 JAN 2002
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2002 AMERICAN CHEMICAL SOCIETY (ACS)

=> s Calcineurin A

L1          1282 CALCINEURIN A

=> s Calcineurin B

L2           503 CALCINEURIN B

=> s l1 and l2

L3           220 L1 AND L2

=> s l3 and fusion

L4            14 L3 AND FUSION

=> s l3 and (LexA or Gal4 or p65 or VP16 or AP)

L5             7 L3 AND (LEXA OR GAL4 OR P65 OR VP16 OR AP)

=> s l4 and l5

L6             1 L4 AND L5
```

WEST

Help | Logout | Interrupt

Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences

Search Results -

Terms	Documents
Calcineurin and fusion	153

Database: US Patents Full-Text Database
US Pre-Grant Publication Full-Text Database
JPO Abstracts Database
EPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Refine Search: Calcineurin and fusion

Clear

Search History

Today's Date: 1/11/2002

DB Name	Query	Hit Count	Set Name
USPT	Calcineurin and fusion	153	L6
USPT	Calcineurin and transgenic	68	L5
USPT	Calcineurin-b	1	L4
USPT	Calcineurin-A	1	L3
USPT	Calcineurin	301	L2
USPT	Calcineurin adj A	0	L1

L6 ANSWER 5 OF 7 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 96106941 MEDLINE
DOCUMENT NUMBER: 96106941 PubMed ID: 8535159
TITLE: Reconstitution of active human calcineurin from recombinant subunits expressed in bacteria.
AUTHOR: Rokosz L L; O'Keefe S J; Parsons J N; Cameron P M; Burbaum J J
CORPORATE SOURCE: Department of Biophysical Chemistry, Merck Research Laboratories, Rahway, New Jersey 07065-9500, USA.
SOURCE: PROTEIN EXPRESSION AND PURIFICATION, (1995 Oct) 6 (5) 655-64.
Journal code: BJV; 9101496. ISSN: 1046-5928.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199602
ENTRY DATE: Entered STN: 19960221
Last Updated on STN: 19980206
Entered Medline: 19960208

AB **Calcineurin**, a protein phosphatase found in eukaryotic cells, presents a challenging problem in heterologous protein expression because it is both heterodimeric and posttranslationally modified. In this paper, we describe the cloning of both subunits (catalytic A and regulatory B) of calcineurin from a human cDNA library and their expression at high levels in Escherichia coli. The **calcineurin A** subunit is expressed as an insoluble glutathione S-transferase **fusion** protein, while the **calcineurin B** subunit is soluble upon direct expression. Catalytically active holoenzyme is derived from the separately expressed subunits using a three-step refolding protocol. First, the **fusion** protein is solubilized, then it is cleaved at the **fusion** junction with thrombin, and, finally, a catalytically competent **calcineurin A**: **calcineurin B**:calmodulin complex is reconstituted by cofolding the separately purified components. In addition, we show that a similar refolding protocol can be applied to a C-terminally truncated form of **calcineurin A**, which lacks an autoinhibitory and calmodulin-binding domain.

L6 ANSWER 4 OF 7 MEDLINE
ACCESSION NUMBER: 96011845 MEDLINE
DOCUMENT NUMBER: 96011845 PubMed ID: 7488022
TITLE: Only in the presence of immunophilins can cyclosporin and FK506 disrupt in vivo binding of **calcineurin A** to its autoinhibitory domain yet strengthen interaction between **calcineurin A** and B subunits.
AUTHOR: Chaudhuri B; Stephan C
CORPORATE SOURCE: Department of Core Drug Discovery Technologies (CDDT), Ciba-Geigy AG, Switzerland.
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1995 Oct 13) 215 (2) 781-90.
Journal code: 9Y8; 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199511
ENTRY DATE: Entered STN: 19960124
Last Updated on STN: 19980206
Entered Medline: 19951124

AB The two immunosuppressants cyclosporin A (CsA) and FK506 exert their major therapeutic effect by inhibiting T-cell activation. It is believed that the drugs first bind to their cellular receptors, known as immunophilins, and then target the protein phosphatase calcineurin for inhibition. The catalytic activity of calcineurin is regulated by its autoinhibitory domain (AID) and by the calcium-binding protein **calcineurin B** (CnB) and calmodulin. We have used the yeast two-hybrid system to show that AID, CnB and calmodulin can only bind to a truncated catalytic subunit of yeast calcineurin (i.e., CnA1 delta), devoid of AID, but not to full-length CnA1. Both CsA and FK506 cause disruption of the CnA1 delta-AID interaction, whereas their presence permits CnA1 delta to bind more strongly to CnB. In contrast, the binding of CnA1 delta to calmodulin is unaffected by the immunosuppressants. Significantly, in the absence of its cognate cytosolic receptor, neither CsA nor FK506 inhibits or stimulates the CnA1 delta-AID, CnA1 delta-CnB interactions. These in vivo observations not only provide supportive evidence for the mechanism by which drug-receptor complexes could modulate calcineurin activity but also unveil the possibility of identifying novel immunophilin-independent calcineurin inhibitors which may disturb the association of CnA1 delta to AID.